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The contribution of causes of death to socioeconomic inequalities in child mortality: New Zealand 1981–1999

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Abstract

Background Socioeconomic inequalities in all-cause child mortality exist in New Zealand; however the inequalities in cause-specific mortality have not been examined. This study examines child mortality inequality by household income between 1981 and 1999, by cause of death.

Methods Data was used from a record linkage study of census and mortality records of all New Zealand children aged 0–14 years on census night 1981, 1986, 1991, 1996 followed up for 3 years for specific causes of mortality between ages 1–14 years. All cohorts were combined to calculate mortality rates, rate ratios, and rates differences for each cause of death.

Results Socioeconomic differences in child mortality (low income compared to high income) were observed for injury (non road traffic) (RR 1.87, 1.35 to 2.58), road traffic injury (RR 1.36, 1.01 to 1.82), and 'other' causes of death (RR 1.81, 1.32 to 2.47). 'Other' and non-road traffic injury deaths together contributed 70% of the total gap in child mortality between the rich and the poor.

Conclusions Socioeconomic differences existed across most broad causes of child death. The major contributors to mortality inequality are diverse, suggesting that the similar distal causes of inequality (e.g. poverty) play out through a myriad of proximal causes. Fortunately there appears to be some scope for policymakers to modify some of the proximal and distal causes of these inequalities.

Summary Statistics New Zealand Security Statement—The New Zealand Census Mortality Study (NZCMS) is a study of the relationship between socioeconomic factors and mortality in New Zealand, based on the integration of anonymised population census data from Statistics New Zealand and mortality data from the New Zealand Health Information Service. The project was approved by Statistics New Zealand as a Data Laboratory project under the Microdata Access Protocols in 1997. The data-sets created by the integration process are covered by the Statistics Act and can be used for statistical purposes only. Only approved researchers who have signed Statistics New Zealand's declaration of secrecy can access the integrated data in the Data Laboratory. (A full security statement is in a technical report at http://www.wnmeds.ac.New Zealand/nzcms-info.html.) For further information about confidentiality matters in regard to this study please contact Statistics New Zealand.

The existence of socioeconomic inequalities in all-cause child mortality in developed countries, including New Zealand, is well described.[1–7] All-cause mortality includes a diverse range of causes of child mortality with dissimilar aetiologies. Despite this apparent diversity, socioeconomic gradients have been reported internationally in almost all of the common causes of child mortality. For example, socioeconomic gradients have been demonstrated in motor vehicle deaths,[5,8,9] child pedestrian injury deaths,[10] fire deaths,[5,6,10] drowning,[5,6] mortality from congenital conditions,[5–7] sudden infant death syndrome,[8,11] and cancer.[5,7,9]

This paper describes socioeconomic inequalities by cause of death groupings in New Zealand children between 1981 and 1999, and assesses which causes of death contribute most to inequality in all-cause child mortality. We then examine the pathways through which parental socioeconomic position (SEP) may be embodied in health, and expressed as child mortality.[12,13] While much of the latter discussion is theoretical (and possibly reductionist due to the complexity of pathways) it attempts to move beyond simple description, and aims to consider reasons.

Methods

The data in this study came from the New Zealand Census-Mortality Study.[14–17] Four population cohorts were constructed by anonymously and probabilistically linking individual census and mortality records over four time periods from 1981 to 1996, inclusive.[15,17] New Zealand Health Information Service provided mortality data for 0–14 year olds for the periods 1981–84, 1986–89, 1991–94, and 1996–99. Four cohorts were created, following children aged 0–14 years on census night for 3 years, with analysis being conducted on those deaths that occurred in children aged 1–14 years. (Note that this study is not well suited to the study of infant mortality due to being a closed cohort.)

The percentage of eligible mortality records linked ranged from 66%–71% in each of the four census cohorts, and the percentage of those links estimated to be correct links was in excess of 96%.[16,18] Linkage varied by age, rurality, ethnicity, and small area deprivation—so linkage weights were applied to overcome any potential misclassification bias of mortality outcome caused by differential success of linkage.[16] For example, if 20 out of 30 deaths in one strata of sex, age, ethnicity and cause of death were linked, then a weight of 30/20 = 1.5 was applied to those 20 linked pairs. Non-linked census respondents were then weighted down slightly to ensure that the total weighted number of children in the cohorts equalled the census night population. Sensitivity analyses published elsewhere suggest these weights work well to adjust for (any) linkage bias.[16]

To be included in the analysis, children must have been at their usual residence on census night, which had to be a private dwelling. All family types were included in the analysis. However an adult over the age of 16, who was also in their usual residence, had to be present on census night. These restrictions resulted in the exclusion of 7–9% of children in each cohort.

The 'exposure' (socioeconomic position) was measured using household income. When income was available on all adults in the house, it was collated and equivalised for household size using the New Zealand-specific Jensen equivalisation index.[14,19] The equivalisation process adjusts for the number of adults and children in each household, recognising that larger families require more income to have the same standard of living as smaller families. Incomes were consumer price index adjusted to 1996 and then attached to each child in the household. Children were divided into three equal-sized income groups, with cut points of low (<NZ$20,600), medium (≥NZ$20,600 to ≤NZ$33,000), and high (>NZ$33,000).

The 'outcome' (cause of death) was divided into 6 groups: road traffic crash (RTC) (ICD9 E810-825), other injury (ICD E800-809, 826-929), cancer (ICD-140-209), congenital (ICD-740-759), suicide/homicide (ICD E950-999), and 'other' (all remaining ICD codes). The three most common causes of death in 'other' were communicable diseases, asthma, and respiratory infection. Pooled results are presented for all cohorts combined, as the purpose of this paper was to assess the socioeconomic gradient by cause of death, not assess any change over time.

Standardised rates, rate ratios, rate differences, and 95% confidence intervals were calculated across levels of income,[20] using the age and ethnic group composition of the 1991 NZ census population as the external standard. Results were standardised by ethnicity, as: ethnicity is a strong determinant of socioeconomic position; ethnicity is also a strong determinant of health independent of socioeconomic position; and the ethnic composition of New Zealand children changed over this period.

The number of children identified as Maori or Pacific increased by 20.7% and 45% respectively, compared to a 13% decline in non-Maori/non-Pacific children between 1981 and 1999. Results are presented for both sexes together to maximise statistical power and because it is not possible for sex to confound the relationship between SEP and child mortality (i.e. whilst the child's sex predicts child mortality, it is not associated with household measures of SEP).

The programme of work of the New Zealand Census Mortality Study has approval from the Wellington Ethics Committee (Reference number 98/7).

Results

Over the cohorts under study there were 2466 (weighted) deaths in children aged 1–14 years. The person years in each income group over the period was 2,329,754 in the low income group; 2,296,849 in the medium income group; and 2,210,060 years in the high-income group. Seven to 9% of all children were excluded from each cohort because they or their parent/caregiver were not at home on census night and an additional 1,406,383 person years (approximately 20% of children) were not available for this analysis due to missing income information.

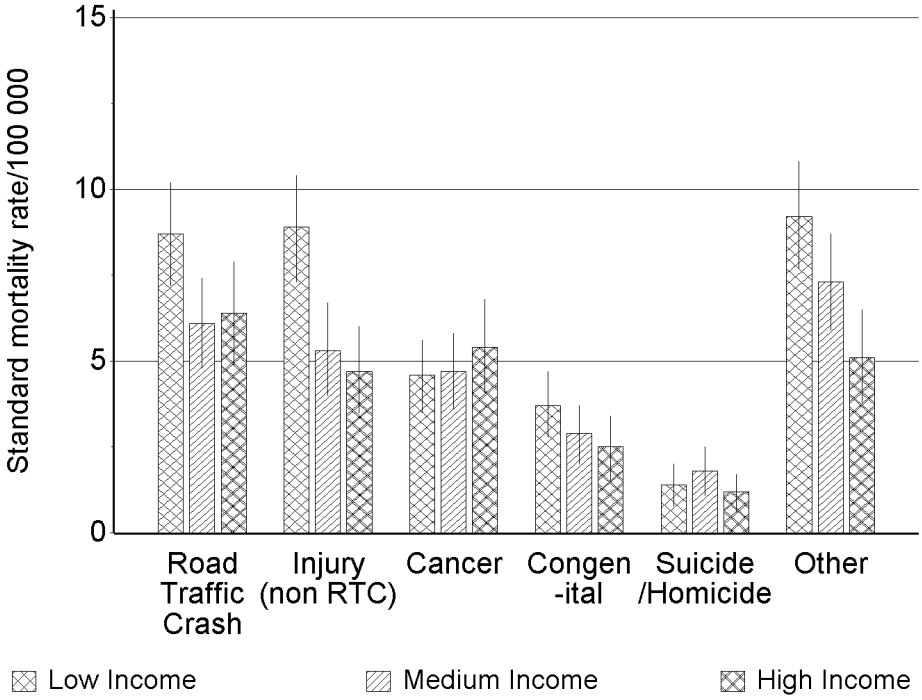
The numbers of deaths; age and ethnicity-standardised mortality rates; rate ratios; and rate differences by cause of death are presented in Table 1 and Figure 2. Both show that socioeconomic gradients in child mortality were seen in all causes of death except cancer. Gradients were most strongly seen in injury (non road traffic), followed by 'other' causes of death and road traffic injuries. Point estimates suggested socioeconomic gradients for both suicide/homicide and mortality from congenital causes. However the confidence intervals included 1 for both of these causes of death. Cancer mortality was the notable exception with a pattern of increasing mortality with higher income is seen, although this was non-significant.

Table 1. Number of deaths in children aged 1-14 years; and standardised (Std) rates, rate ratios (SRR), and rate differences (SRD) by equivalised household income (1981–1999)

Cause of Death	Income	Deaths	Std Rate	95%CI	SRR	95%CI	SRD	95%CI
All Cause	Low	834	36.5	(33.4 – 39.5)	1.44	(1.25 – 1.66)	11.1	(6.9 – 15.4)
	Medium	609	28.2	(25.4 – 30.9)	1.11	(0.95 – 1.29)	2.8	(-1.2 – 6.9)
	High	519	25.3	(22.4 – 28.3)	1.00		0.0	
Road Traffic Crash (RTC)	Low	207	8.7	(7.2 – 10.2)	1.36	(1.01 – 1.82)	2.3	(0.1 – 4.4)
	Medium	132	6.1	(4.8 – 7.4)	0.96	(0.70 – 1.32)	–0.3	(-2.3 – 1.7)
	High	132	6.4	(4.9 – 7.9)	1.00		0.0	
Injury (non-RTC)	Low	198	8.9	7.3 – 10.4	1.87	1.35 – 2.58	4.1	2.1 – 6.2
	Medium	111	5.3	(4.0 – 6.7)	1.13	(0.78 – 1.62)	0.6	(-1.2 – 2.4)
	High	102	4.7	(3.5 – 6.0)	1.00		0.0	
Cancer	Low	105	4.6	(3.5 – 5.6)	0.84	(0.60 – 1.17)	–0.9	(-2.6 – 0.8)
	Medium	105	4.7	(3.6 – 5.8)	0.87	(0.62 – 1.22)	–0.7	(-2.4 – 1.0)
	High	108	5.4	(4.1 – 6.8)	1.00		0.0	
Congenital	Low	87	3.7	(2.8 – 4.7)	1.52	(0.95 – 2.43)	1.3	(-0.1 – 2.6)
	Medium	60	2.9	(2.0 – 3.7)	1.17	(0.72 – 1.91)	0.4	(-0.9 – 1.7)
	High	45	2.5	(1.5 – 3.4)	1.00		0.0	
Suicide/Homicide	Low	33	1.4	(0.8 – 2.0)	1.17	(0.62 – 2.22)	0.2	(-0.6 – 1.0)
	Medium	39	1.8	(1.1 – 2.5)	1.53	(0.82 – 2.85)	0.6	(-0.3 – 1.5)
	High	27	1.2	(0.6 – 1.7)	1.00		0.0	
Other	Low	207	9.2	(7.7 – 10.8)	1.81	(1.32 – 2.47)	4.1	(2.1 – 6.2)
	Medium	159	7.3	(5.9 – 8.7)	1.42	(1.03 – 1.97)	2.2	(0.2 – 4.1)
	High	105	5.1	(3.8 – 6.5)	1.00		0.0	

Deaths are weighted deaths, rates are age and sex standardised and per 100,000, SRD are per 100,000.

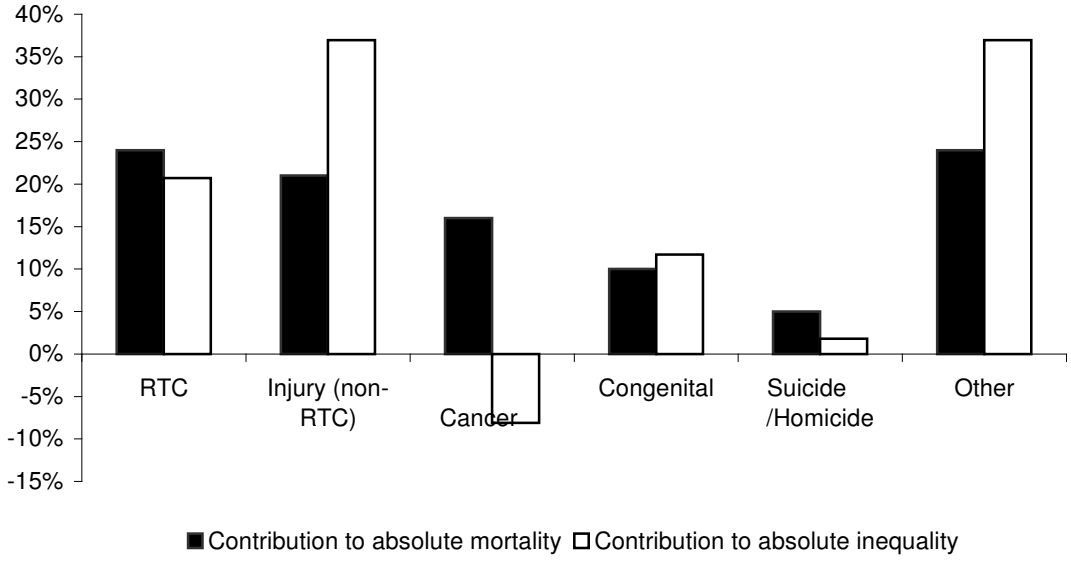
Figure 1. Mortality rates for various causes of death among children aged 1–14 by equivalised household income (1981–1999)



Error bars=95% confidence intervals.

illustrates the contribution of different causes of death to overall mortality and to absolute inequality. It demonstrates that the causes of death that contribute proportionately most to child mortality (road traffic injuries and 'other' causes of death) are not the same as the causes of death that contribute most to inequality. Indeed 'other' causes of death and non-road traffic injury contribute approximately 70% of the absolute inequality in child mortality. (These percentages were calculated by dividing the standard rate difference for the low-income group, for each cause of death, by the standard rate difference for the low-income group in all-cause mortality.)

Figure 2. Contribution to total mortality and absolute inequality by cause of death



Discussion

This study illustrates the inequitable distribution of child mortality in New Zealand, with socioeconomic gradients seen in most common causes of child mortality in New Zealand, despite often diverse aetiological pathways. The contribution of two very dissimilar causes of death to 70% of absolute inequality suggests some commonality of process, which leads to the outcome of child mortality.

The large study size of the combined New Zealand Census-Mortality Study cohorts allows reasonably precise estimates of cause-specific socioeconomic gradients in child mortality. However, despite the use of an entire population sample, some causes of death were simply too uncommon in New Zealand children to allow exact determination of gradients, meaning within each group there are a number of causes of death, with differing aetiology. There will also be some imprecision around the percentage contribution of differing causes of death to absolute inequality, but we are confident that this will not alter the broad findings.

Similar socioeconomic gradients were seen by maternal education (data not presented). This suggests that selection bias is not the explanation for the observed gradients (data were available for nearly all children on the educational level of their mothers). Moreover, the findings in this study are unlikely to be due to health selection (whereby parents with children who die are likely to become socioeconomically deprived in the period before death, thus 'appearing' to be of low socioeconomic position) as the most common cause of child mortality is injury, an unanticipated acute event.

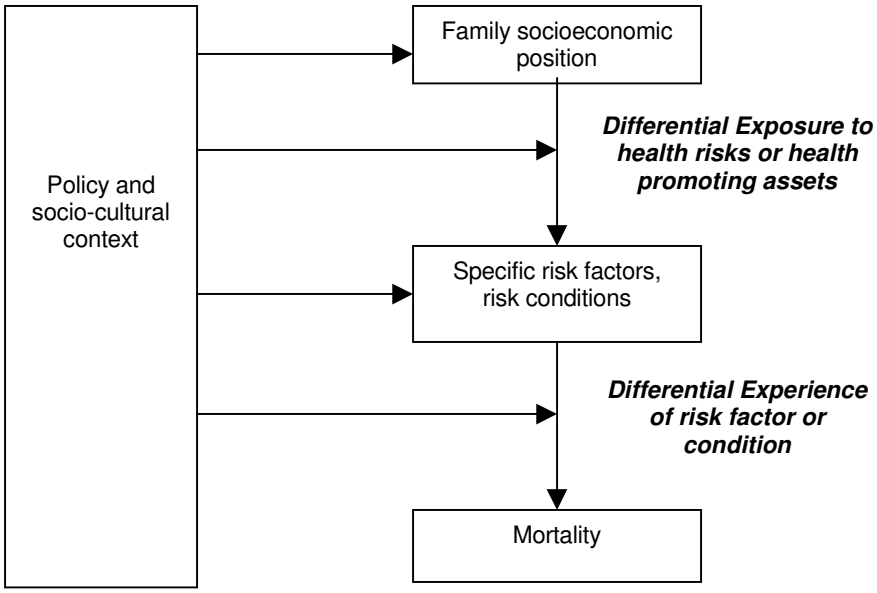
The socioeconomic gradients by household income shown here will be confounded by other socioeconomic factors, but the intention of this paper is not to look at the

independent effect of income. Rather we use income to represent the process of social stratification of child mortality that occurs within a society.

This study also confirms that excess mortality risk is not simply a poverty-related phenomenon in New Zealand; children in the middle-income group had mortality rates that were, in the most part, higher than those in the high-income group for a number of causes of death. This has important implications for the type of public health interventions that will be required to reduce inequalities; programmes targeting children in low-income households will miss the opportunity to prevent excess mortality in middle-income children.

Figure 3 illustrates one explanatory model that can be used to consider how these socioeconomic gradients are generated and how diverse causes of death can show such similar patterns. This is a modified version of the framework proposed by Diderichson and Hallqvist (cited in Laflamme 2000[21]). This model suggests that family SEP exposes children differentially to either specific risk(s) or health promoting assets, which avert the risk. Children may also have a different *experience* of this particular risk or disease depending on family socioeconomic position. This model highlights that the wider context of policy and socio-cultural environment may influence all of these layers, family socioeconomic position, and the exposure and experience of risk. The remainder of the discussion focuses on applying this model to causes of child mortality.

Figure 3. Model of the socioeconomic determinants of fatal injury



Source: Laflamme 2000

Road traffic mortality—Taking road traffic injuries as an example, within this group there are two main types of deaths, child pedestrian deaths, and motor vehicle passenger deaths. The aetiology of child pedestrian deaths is well studied; Table 2

illustrates the differing exposures that children of different socioeconomic groups have to both health injurious, as well as health-promoting resources.

Table 2. Mechanisms by which differential exposure to child pedestrian risk could occur

Poor children have increased exposure to:	Rich children have increased exposure to
• High traffic flow in neighbourhood[22] • Fast traffic speeds in neighbourhood[22,23] • High density parking in neighbourhood[22,23] • Walking to school[24] • Higher number of roads crossed while walking to school[25,26]	• Parental or adult supervision while walking to or from school[25,27] • Safe places to play[28] • Car ownership[25] • Fenced driveways[29]

Note: References 22, 24, 25, 27, and 29 are New Zealand research.

The other main cause of mortality in this group is vehicle crashes in which children are passengers. International evidence mostly finds a socioeconomic gradient,[6,10,30] but the precise pathways from SEP to injury are less well studied. It seems likely that children in lower socioeconomic groups are more likely to be in cars that are less crash-worthy (in both design and lack of maintenance). Children of lower socioeconomic groups may also be more likely to be in vehicle crashes through being in cars with drivers more predisposed to crash.[31]

Factors subsequent to the event could also be important in generating socioeconomic gradients in mortality (i.e. differential experience of the risk described in the model in Figure 3). For example, children in lower socioeconomic groups (as measured by type of car driven) are less likely to be restrained by either car seats or belts,[32] thereby being more susceptible to severe injury or mortality in the event of a crash. There is some research suggesting that, in adults, obesity is associated with increased mortality after a car crash.[33] This has not been researched in children, but is worth consideration given the strong association between lower SEP and obesity in New Zealand children.[34] Car factors may also play a role, as children in higher socioeconomic groups may be less likely to sustain severe or life threatening injuries thanks to features such as airbags, and intrusion bars.

Non-road traffic injury mortality—The most common causes of mortality in the non-road traffic injury category in 1-14 year olds in New Zealand are drowning and fire deaths.[35] While this study was not able to look at these specific causes, international evidence supports the existence of socioeconomic differences in both drowning and fire mortality.[5,6,10]

Risk factors associated with fires, such as poor housing conditions and parental smoking, cluster in poorer households.[36] Moreover, children in households of higher SEP are more likely to have access to smoke alarms and telephones, both of which reduce risk of fire deaths.[36,37]

Possible reasons for socioeconomic gradients in drowning include differing exposure to pools, differences in safety aspects around water such as parental supervision and pool fencing, and differing experience of water (studies in both New Zealand and

Australia suggest that children in lower socioeconomic groups have poorer swimming skills compared to their peers).[38,39]

'Other' mortality—The group of 'other' is a heterogeneous group of mortality causes, with the largest causes of death being communicable diseases, asthma, and respiratory infections. There is evidence that risk factors for infectious diseases such as meningococcal disease and pneumonia cluster in more deprived households.[40,41] Given the strength of the gradients observed in this study, the specific causes of death need to be studied more closely, in order to determine where differential exposure to (and experience of) risk by SEP occurs.

Cancer mortality—Similar to most other international studies,[42–46] this study found no evidence of socioeconomic gradients in child cancer mortality. (An association between increasing cancer mortality and decreasing SEP was previously reported for just the 1991-94 cohort,[9] however it would appear that this was a chance finding.) This is in contrast to adults in New Zealand in whom cancer is increasingly patterned by SEP.[47]

Congenital mortality—The findings in this study provide some support for the association described by others between SEP and mortality from congenital conditions,[5–7,48] although understanding this relationship is complicated by the heterogeneous nature of congenital abnormalities and the multiple factors that influence mortality outcome (incidence, prevalence and case fatality). The differential distribution of risk factors for congenital anomalies such as low folic acid intake,[49,50] obesity,[50] and proximity to landfills,[51] could contribute to the observed socioeconomic gradients from congenital causes of mortality.

Suicide/homicide mortality—The lack of any association between suicide and homicide deaths with SEP was somewhat surprising, as there is a strong relationship between suicide and lower SEP for adults in New Zealand.[52] Evidence from the USA shows a relationship between poverty and child homicide,[5,6] and there is some evidence to suggest that risk factors for these types of death are disproportionately placed in lower socioeconomic households.[53] However, our New Zealand findings were based on a small number of deaths, reflected by the wide confidence intervals.

To understand the process of how socioeconomic inequalities lead to health outcomes, this discussion has been framed around exposure to risk and risk-experience. These are relatively proximal risk factors; the experience of risk factors for mortality are determined by more distal mechanisms, for example transport policy, determinants of income, availability of food and national policy on folic acid fortification.

In conclusion, intervening to reduce the child mortality inequalities in New Zealand shown in this paper should be a key priority for government. This study has shown that disease-related mortality and non-road traffic injury mortality are the largest contributors to child mortality inequalities in New Zealand. Addressing these issues needs to be given priority; this includes further research into inequalities in child injury morbidity and also into interventions that reduce inequalities. In addition, the influence of existing and future policy needs to be evaluated carefully for potential effects on the determinants of child health and mortality.

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